IN THE CLAIMS:

Kindly cancel claims 3-9, 11 and 14-19 without prejudice or disclaimer.

Kindly enter the following amended claims:

1. (Twice Amended) An isolated polypeptide belonging to a subfamily of the Immunoglobulin Superfamily consisting essentially of all or part of the amino acid sequence of murine Confluency Regulated Adhesion Molecule 1 (muCRAM-1, SEQ ID NO: 13), the isolated polypeptide being capable of modulating vascular endothelium function.

2. (Twice Amended) An isolated polypeptide belonging to a subfamily of the Immunoglobulin Superfamily consisting essentially of all or part of the amino acid sequence of human Confluency Regulated Adhesion Molecule 1 (huCRAM-1, SEQ ID NO.: 15), the isolated polypeptide being capable of modulating vascular endothelium function.

10. (Amended) The isolated polypeptide according to claim 13, wherein the polypeptide is a soluble polypeptide that inhibits transendothelial migration of leukocytes.

12. (Twice Amended) The isolated peptide according to claim 10, the peptide comprising at least one sequence against which anti-CRAM antibodies can be directed, the at least one sequence being selected from the group consisting of extracellular domain V, extracellular domain C_2 and the membrane proximal cytoplasmic sequence defined by amino acids 266-272 of SEQ ID NO.: 13.

13. The isolated polypeptide as claimed in claims 1, or 2 in soluble form.

Kindly enter the following new claims.

20. (New) The isolated polypeptide according to claim 13, wherein the isolated soluble polypeptide is capable of modulating vascular permeability.

21. (New) An isolated, soluble polypeptide belonging to a subfamily of the Immunoglobulin Superfamily having essentially 100% sequence homology with the amino acid sequence of muCRAM-1, set forth in SEQ ID NO: 13, or having essentially 100% sequence homology with the amino acid sequence of human huCRAM-1, set forth in SEQ ID NO.: 15;

wherein the isolated polypeptide polypeptide exhibits at least one function selected from the group consisting of inhibition of transendothelial migration of leukocytes and modulation of vascular permeability.